

## Case Report

# Aphonia due to vocal cord impairment induced by carbon monoxide poisoning

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**Case:** We describe a case of a 22-year-old woman with alteration in consciousness and aphonia due to vocal cord impairment after carbon monoxide exposure. Brain magnetic resonance imaging revealed high signal intensity in bilateral globus pallidus and the pars reticulata of the substantia nigra on T2- and diffusion-weighted images. Laryngeal fiberoscopy showed bilateral immobilization of the vocal cords in median position during both inspiration and phonation. Although the effects of hyperbaric oxygen therapy remain ambiguous, these symptoms and magnetic resonance imaging findings subsided.

**Outcome:** Aphonia due to vocal cord impairment, as a presenting symptom of carbon monoxide poisoning, has not been previously reported. We considered the cause of aphonia was vocal cord abductor paralysis or dystonia of intralaryngeal muscles after the carbon monoxide exposure.

**Conclusion:** Even though aphonia is an unusual symptom in a patient with carbon monoxide poisoning, it must be taken into consideration.

**Key words:** Dystonia of intralaryngeal muscles, pallidoreticular damage, parkinsonian features, vocal cord abductor paralysis

## INTRODUCTION

CARBON MONOXIDE (CO) poisoning causes various neurological symptoms including memory impairment, consciousness disturbance, and parkinsonian symptoms.<sup>1–3</sup> However, aphonia, as a presenting symptom of CO poisoning, has not been previously reported. In this report, we describe the case of a CO-poisoned patient presenting with aphonia due to vocal cord (VC) impairment after CO exposure.

## CASE REPORT

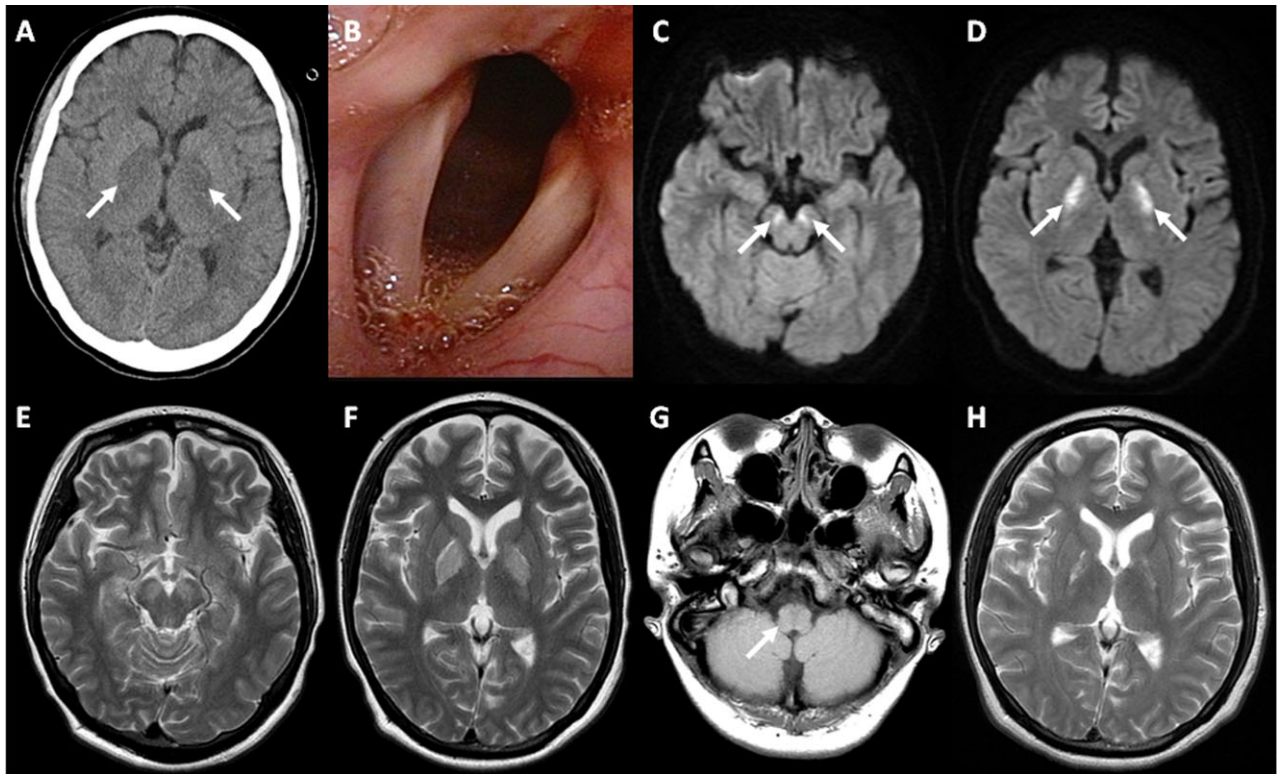
A 22-YEAR-OLD, PREVIOUSLY HEALTHY Japanese woman was found unconscious in her parked car. As

burned briquettes were found inside the car, CO poisoning due to a suicide attempt was suspected, and she was transferred to our hospital. On admission, 1 h after the patient had been found, the patient showed a deep coma state (Glasgow Coma Scale [GCS], E1VTM1); she had been intubated before arriving at our hospital. The computed tomography scan of the head showed a low density area of bilateral globus pallidus (GP) (Fig. 1A). The urine toxicology screening (phencyclidine, benzodiazepine, cocaine, antihypnotic agents, marijuana, morphine, barbiturates, and tricyclic antidepressants) was negative. Arterial blood gas examination carried out 1 h after ventilation with 100% oxygen revealed a carboxyhemoglobin level of 11.4%; the patient was given a diagnosis of CO poisoning. Hyperbaric oxygen therapy (HBO) at 2.0 atmosphere absolute for 90 min was administered. After the first HBO, the carboxyhemoglobin level decreased to less than 1% and the patient was admitted to an intensive care unit. We carried out HBO every weekday.

The patient's state of consciousness improved on day 3 (GCS, E3VTM6) and she was extubated on day 5. After extubation, the patient had loss of voice and severe difficulty swallowing, but was able to read and write. Her Mini Mental State Examination score was 23; slight disorientation, dyscalculia, and disturbance of attention were observed. The

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**Fig. 1.** Computed tomography scan of the head on day 1, laryngeal fiberscopy on day 10, and brain magnetic resonance imaging on days 11 and 45 in a 22-year-old woman with aphonia after carbon monoxide exposure. The scan of the head shows a low density area in the bilateral globus pallidus (GP) (A, arrows). Laryngeal fiberscopy reveals bilateral immobilization of the vocal cords in median position during phonation (B). Diffusion-weighted images obtained 11 days after carbon monoxide exposure show symmetrical high signal intensity lesions in the bilateral substantia nigra (C, arrows) and in the GP (D, arrows). T2-weighted images also reveal symmetrical high signal intensity lesions in the bilateral substantia nigra (E) and in the GP (F). There is no alteration in the medulla oblongata and corticobulbar tracts on T2-weighted image (G, arrow). T2-weighted images on day 45 show atrophied slightly high signal intensity lesions in the GP (H).

laryngeal fiberscopy revealed bilateral immobilization of the VC in median position during both inspiration and phonation (Fig. 1B) and decreased sensation of the VC. No evidence of traumatic injury, edematous lesions, or arytenoid cartilage dislocation was observed. Therefore, a brain lesion was suspected rather than peripheral recurrent nerve paralysis due to tracheal intubation. On day 11, brain magnetic resonance imaging (MRI) revealed high signal intensity in the bilateral GP and the pars reticulata of the substantia nigra on T2- and diffusion-weighted images (Fig. 1C–G). At the neurological examination, the patient showed an improved level of consciousness (GCS, E4V1M6). Parkinsonian symptoms such as tremor, muscle rigidity, or akinesia were not observed.

The patient was able to speak from day 36, so HBO ceased. Her consciousness and cognitive function improved gradually and her Mini Mental State Examination score was 26 on day 36. The difficulty swallowing also improved and

she initiated oral ingestion on day 42. On day 45, a follow-up brain MRI revealed improvement, with the T2-weighted image showing isosignal intensity in the bilateral substantia nigra and slightly high signal intensity lesions in the GP (Fig. 1H). She was discharged on day 57 with mild cognitive impairment.

## DISCUSSION

THE MOST DISCRIMINATIVE point in this study is that the CO-poisoned patient presented with aphonia due to VC impairment. Anatomically, the aphonia due to VC impairment could be caused by lesions of the cerebral cortex, basal ganglia, or brainstem nuclei. In this case, brain MRI showed no alteration in the medulla oblongata, corticobulbar tracts, or cerebral cortex. Therefore, we suspected a lesion of the basal ganglia as responsible for this VC

impairment. Recently, a case of aphonia induced by simultaneous bilateral ischemic infarctions of the putamen nuclei was reported.<sup>4</sup> The putamen receives projections from cortical motor areas and sends efferent projections to the internal segment of the GP, which sends efferent projections to the ventrolateral nucleus of the thalamus and closes the loop by feedback to the cortex.<sup>4</sup> Thus, we considered that bilateral lesions of the GP in this case interrupted the flow of impulses from cortical areas to the thalamus, resulting in VC paralysis.

As a complementary hypothesis, the mechanism of this VC impairment is thought to be a VC abductor paralysis or dystonia of intralaryngeal muscles as a parkinsonian symptom. Patients with multiple system atrophy, a neurodegenerative disorder characterized by parkinsonian symptoms and autonomic dysfunction, sometimes present with respiratory failure and laryngeal stridor.<sup>5–8</sup> The cause of these symptoms is not fully understood, but it is thought to be caused by VC abductor paralysis<sup>5,6</sup> or dystonia of intralaryngeal muscles.<sup>7,8</sup> An electromyogram of the intralaryngeal muscles would have been helpful to distinguish between two possibilities,<sup>9</sup> but we could not perform it in this case. Taken together, the interruption of feedback loops in basal ganglia may result in the VC impairment as a parkinsonian symptom.

A potential weakness of our study is that we cannot exclude the possibility of other etiologies. As described above, immobilization of the VC was observed bilaterally and traumatic changes were not observed by the laryngeal fiberoptic. Moreover, peripheral recurrent nerve paralysis due to tracheal intubation generally occurs unilaterally.<sup>10</sup> Therefore, we conjectured that the cause of bilateral VC immobilization was a central lesion rather than peripheral recurrent nerve paralysis. The findings of laryngeal fiberoptic also ruled out psychological aphonia because the immobilization of VC was observed even during coughing.

## CONCLUSION

**I**N SUMMARY, WE described a case of CO poisoning with aphonia due to VC impairment. In comparison to multiple system atrophy, we considered the etiology of aphonia was VC abductor paralysis or dystonia of

intralaryngeal muscles as a parkinsonian symptom caused by bilateral lesions of the GP after CO exposure. Even though aphonia is an unusual symptom in a patient with CO poisoning, it must be taken into consideration.

## CONFLICT OF INTEREST

**N**ONE.

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